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REPORT ON A PRELIMINARY FIELD SURVEY OF THE SO-CALLED "ALKALI DISEASE" OF LIVESTOCK

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INTRODUCTION

For a number of years there has been known, in certain areas of the north-central Great Plains, what is called the "alkali disease" of livestock. This name is clearly a misnomer as it is now certain that the disease is not caused directly or indirectly by alkali water, or by alkali spots in the soil, as was supposed by the early settlers of those sections. The specific cause of the malady is not known, although it is evident that either grain or forage grown in certain areas may cause the disease. As early as May 1931, Henry G. Knight, Chief of the Bureau of Chemistry and Soils, United States Department of Agriculture, suggested that possibly the disease might be caused by traces of selenium absorbed by plants from the soils of the areas in question. While experimental evidence to date is not entirely conclusive on this point, indications are that this suggestion is correct. Since the preparation of this report, considerable progress has been made in the study of various phases of this problem. The results will be published separately by the respective investigators. This circular is restricted to an historical summary and the results of the survey.

The earliest reference to this disease the writers have been able to find is a report by Madison,[1] who described an unusual condition

[1] MADISON, T. C. SANITARY REPORT—FORT RANDALL. *In* Coolidge, R. H., Statistical Report on the Sickness and Mortality in the Army of the United States . . . January 1855 to January 1860. [U.S.] Cong. 36th, 1st Sess., Senate Ex. Doc. 52 : 37–41. 1860.

in Army horses at Fort Randall, Territory of Nebraska,[2] in August 1856. Some of the symptoms described are typical of the disease under discussion. In addition to the public mounts, private horses of the officers, Indian ponies, and a few mules were similarly affected.

In 1904 Peters [3] reported a disease of livestock in northern Nebraska, which had the same symptoms as the so-called "alkali disease." He attributed this disease to a fungus disease of corn caused by a species of *Fusarium*, which was simultaneously described by Sheldon [4] as *Fusarium moniliforme*. It is now well known that this fungus is very common and widely distributed on corn throughout the world wherever corn is grown, and the so-called "alkali disease" of livestock is rather limited in distribution. Therefore, this fungus disease of corn clearly is not the cause of the disease of livestock in question. Peters states that the condition was reproduced in swine by feeding them cultures of the fungus grown on crackers and corn meal but does not mention the source of the corn meal. He states, however, that corn was obtained from farms on which the disease of livestock had occurred. If the corn meal was obtained from corn grown in affected areas, this could explain the results that he reports, entirely aside from the presence of the fungus. From Peters' excellent description of the disease in horses, cattle, swine, and chickens, it is clear that the malady in question is identical with that which is the subject of this report.

In 1921 Lipp [5] briefly described the malady as it occurred in localized areas in South Dakota, chiefly in certain counties bordering the Missouri River.

For a number of years, different departments at the South Dakota State College of Agriculture and Mechanics Arts have made studies of this malady, and field surveys also have been made at different times by the Bureau of Animal Industry of the United States Department of Agriculture. In some of the reports, ergotism has been suspected of being the cause of at least some of the manifestations. However, it is pointed out that the malady occurred during certain years when ergot was rare or absent. Furthermore, corn from the affected areas was one of the principal crops incriminated, and it is well known that ergot does not attack corn. Therefore the malady in question is clearly distinct from ergotism. The trouble is also clearly distinct from the disturbance termed "alkali poisoning", by Miller,[6] which is caused by livestock drinking too abundantly of water that is unusually high in salts, that is, so-called "alkali water." The malady covered by this report also is distinct from the so-called "alkali disease" of livestock in the Pecos Valley of New Mexico and Texas, described by Marsh and Roe,[7] which is caused by poisonous plants.

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[2] Fort Randall is reported as having been located just west of the Missouri River in the Territory of Nebraska, latitude 43°01′ N., longitude 98°12′ W. This location is now included in Gregory County, S.Dak., just north of the Nebraska-South Dakota line.

[3] PETERS, A. T. A FUNGUS DISEASE IN CORN. Nebr. Agr. Expt. Sta. Ann. Rept. 17: 13–22, illus. 1904.

[4] SHELDON, J. L. A CORN MOLD (FUSARIUM MONILIFORME N. SP.). Nebr. Agr. Expt. Sta. Ann. Rept. 17: 23–32, illus. 1904.

[5] LIPP, C. C. ALKALI DISEASE. Vet. Alumni Quart. [Ohio State Univ.] 10: 54–55. 1922.

[6] MILLER, M. R. ALKALI POISONING OF LIVESTOCK. Vet. Med. 21: 268–273. 1926.

[7] MARSH, C. D., and ROE, G. C. THE "ALKALI DISEASE" OF LIVESTOCK IN THE PECOS VALLEY. U.S. Dept. Agr. Circ. 180, 8 pp., illus. 1921.

SCOPE OF THE SURVEY

As a result of experimental work done during the preceding 3 years in the chemistry department of the South Dakota Agricultural Experiment Station, this malady was again brought to the attention of the United States Department of Agriculture the latter part of April 1931. A cooperative preliminary field survey was planned to secure additional first-hand information on the manifestations of the malady, its geographic distribution, possible plant and soil relationships, and the economic importance of the disease. The survey was made by the writers July 18 to August 17, 1931, through the central and southwestern parts of South Dakota, parts of northern Nebraska, and the eastern edge of Wyoming. The malady was

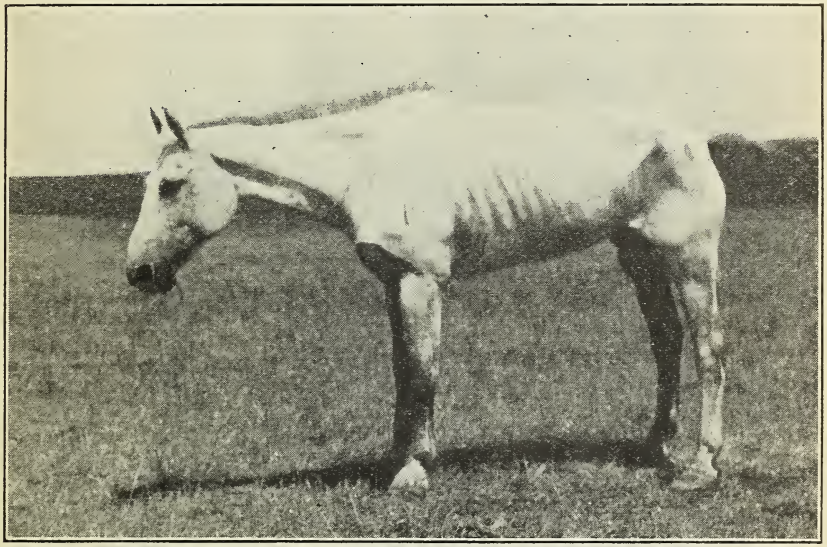

FIGURE 1.—Nine-year-old gelding, severely "alkalied" for 2 years, showing loss of hair from mane and tail.

found locally in these areas on horses, cattle, swine, and poultry. It was also reported on mules. Very few sheep were found in the affected territory, and no dependable information was obtained as to their susceptibility.

OBSERVATIONS ON ANIMALS

In horses, cattle, and swine, the malady manifests itself clinically by an alteration in the growth of the horn of the hoofs and a loss of hair from the mane and tail of horses, and switch of cattle, and the hair of swine (figs. 1–5). There are various gradations of these conditions, from mild cases in which there may be only subnormal growth or a loss of hair, to severe cases in which a break in the continuity of the growth of the walls of the hoofs develops, followed eventually by a sloughing-off of the old hoofs. When this happens,

the animals are more or less lame for months. In the more severe cases, the animals move about with great difficulty (fig. 6) and, unless given careful attention, may die of thirst or starvation. Many of the most severely affected animals die or are destroyed. If taken from "alkalied" areas or given good food, the animals recover, but it is the belief of many that such animals are never quite so valuable as before an attack.

In swine, particularly in young pigs, in addition to lesions produced in the feet and loss of hair, death also may occur. After being

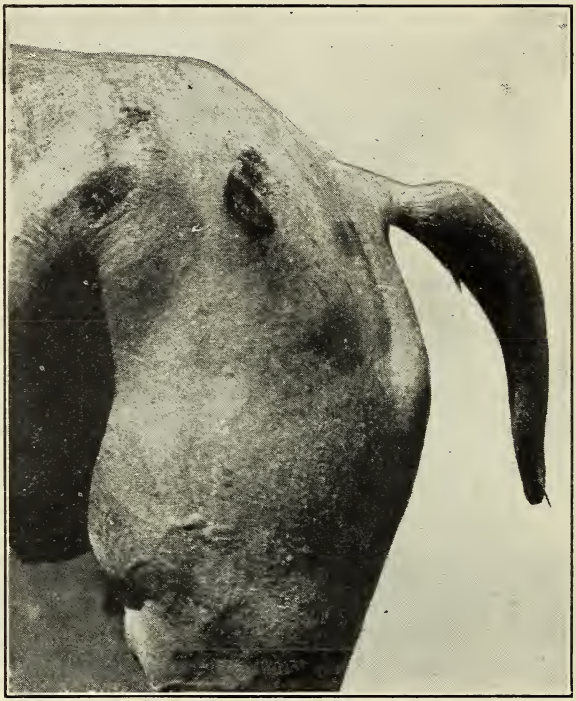

FIGURE 2.—Hind quarter of gelding shown in figure 1, showing distention of stifle joint and loss of hair from tail; also bed sores resulting from being down a great deal of the time on account of sore feet.

badly "alkalied", many of the animals make but little, if any, gain in weight in spite of plenty of nutritious feed.

In the course of the survey, post-mortem examinations were made of a horse and a cow, each of which had been affected at least 2 years. In both of these animals striking internal lesions were found: Erosions on the articular surfaces of many of the joints (fig. 7), edema in the vicinity of the joints, and gelatinous condition of the bone marrow. This indicates the possibility that the casual factor, whatever it may be, affects the entire body.

In poultry the malady manifests itself by the nonhatchability of many of the eggs. The fertility of the eggs may be high, but the few young that hatch are weak and have a "greased" appearance.

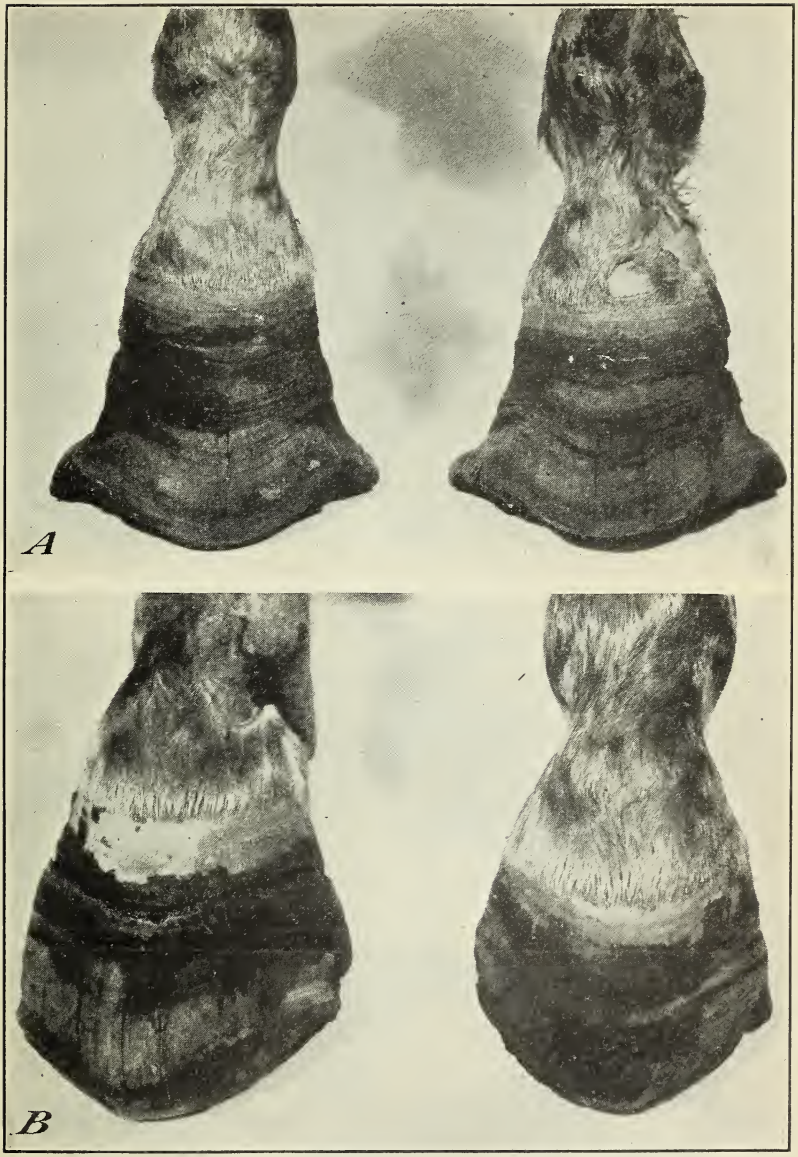

FIGURE 3.—Feet of gelding shown in figure 1, showing (*A*) front feet and (*B*) hind feet.

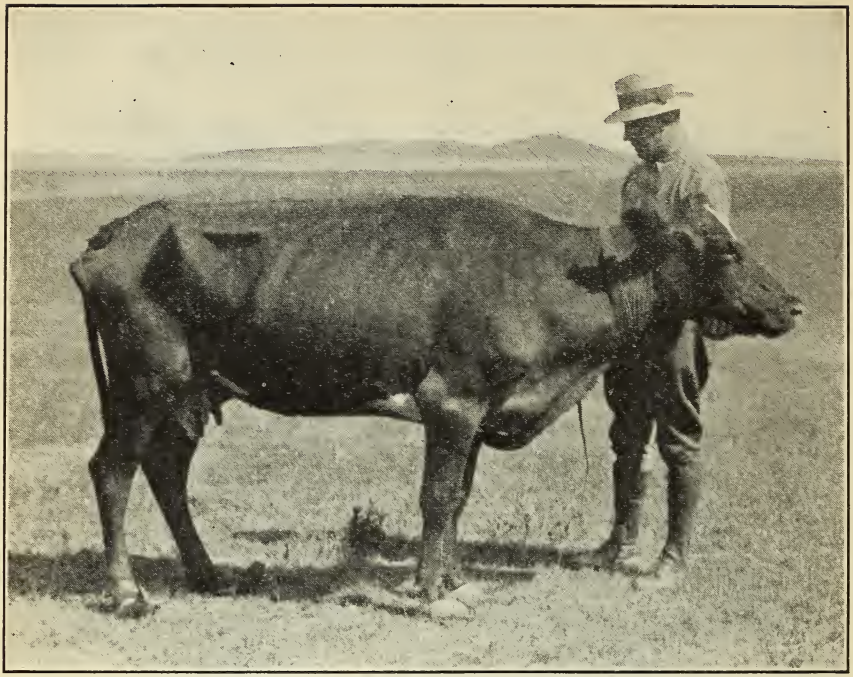

FIGURE 4.—Eight-year-old cow, severely "alkalied" for 2 years.

FIGURE 5.—Feet of cow shown in figure 4, showing abnormal growth of hoofs: (*A*) front feet; (*B*) hind feet.

OBSERVATIONS ON NATIVE FLORA AND CROPS

As far as could be determined by the limited survey made, the native flora of the regions where the malady was found was essentially similar to that of the adjoining areas. Throughout these areas, west-

FIGURE 6.—"Alkalied" steer assuming a kneeling position while grazing, on account of sore feet.

ern wheatgrass (*Agropyron smithii*) was found to be the predominating grass of the lowlands, while *Andropogon*, *Stipa*, *Bouteloua*, and *Aristida* predominated on the highlands, with buffalo grass (*Bulbilis dactyloides*) predominating on the intermediate elevations,

FIGURE 7.—Eroded areas (indicated by arrows) on articular surfaces of the ulna from the cow shown in figure 4.

and various intergradations. Some cordgrass (*Spartina michauxiana*) also was found in the lower draws, and occasionally some saltgrass (*Distichlis spicata*) was noted in low spots, particularly near watering dams. In one of these locations, on the Lower Brule

Indian Reservation, in South Dakota, *D. spicata* was found to be infected with the rust fungus *Puccinia subnitens* Diet. Ergot was almost entirely absent throughout the area, only one ergot sclerotium being found on a single head of *A. smithii.* It was learned, however, that ergot occurs locally in considerable abundance in some of the areas in certain years.

The principal cultivated crops found in the affected areas were wheat, corn, barley, oats, rye, flax, sorghum, millet, and alfalfa. Except for injury from drought and grasshoppers, these crops developed normally. However, grasshoppers did not injure sorghum. The grain from the cereals appeared normal except for shriveling due to drought.

No differences were noticed in the appearance of the native and crop plants in the affected areas as compared with corresponding plants outside of those areas.

OBSERVATIONS ON SOILS

Soil seems to be an important factor in the occurrence of this diseased condition of livestock. Every case investigated occurred on certain soils that were called Pierre clay or Pierre clay loam, in the soil-survey reports, or could be attributed to grain or hay grown on such soils. These soils have developed from the geological formation known as Pierre shale. The Pierre clay, commonly known as " gumbo ", is by far the more extensive in the affected area. On the more level areas the surface 10-inch layer is dark, slaty, olive-brown or almost black, extremely heavy clay. When moderately moist, the soil is finely granular and is mellow and friable, but, when wet, it runs together into a very sticky mass. This surface soil is underlain by a dark olive-brown clay, which changes gradually downward into a light olive-brown, highly calcareous clay. A phase of this soil on slopes and rolling areas, where the greater number of cases were observed, has a lighter-colored olive-brown surface soil, which is calcareous from the surface downward. Masses of gypsum crystals and lime aggregations often occur within 12 inches of the surface, and partly decomposed shale filled with salts, including lime and gypsum, occurs at a depth of less than 3 feet. On these rolling areas the soil is immature, and evidently the constituents of the original shale have not been removed by leaching. On the flat areas some leaching has taken place in the upper layer of the soil, but not in the subsoil.

The diseased condition occurs in a general way over the entire area of these soils that were covered by this survey, but apparently it is not evenly distributed. No reports could be obtained nor cases observed in some areas comprising many square miles.

The survey has not been sufficiently thorough to justify a positive statement that the malady is found on no other soils, but, in the extensive region covered, it could not be attributed to any other soil.

CONCLUSIONS

As a result of this survey it is evident that the toxicity of the grains and grasses grown in some locations is greater than the toxicity of those grown in other locations. Also, there seem to be

variations from season to season. Therefore losses vary with the degree of toxicity of grains and grasses under any given set of conditions. Where the disease is severe, considerable livestock may die or have to be killed, and eggs cannot be used for hatching purposes. In such localities, the raising of hogs or poultry, or both, and even cattle, has been discontinued, and tractor farming of small grain for the market is the sole substitute. Such grain, known to have been produced on affected soil, brings only minimum prices. In two specific cases noted in 1931, the so-called "alkalied" grain brought only half the price of good grain from unaffected farms. Likewise, affected animals usually bring only minimum prices when marketed. Horses that are so severely "alkalied" as to lose their hoofs cannot be worked for months, during which time they require special care. In some cases they become repeatedly "alkalied", so that they cannot be worked for a year or more. Owners of some badly affected farms have abandoned them, chiefly on account of this trouble, not being able to finance, lease, or sell them. In many cases, new renters of affected farms, unfamiliar with the conditions, have had heavy losses within a year after moving to such farms.

At present the only control measures known are: (1) Affected animals should be transferred to feed on unaffected areas; (2) "alkalied" grain, hay, or grass should not be used for feed or pasture; (3) areas definitely known to produce "alkalied" grain and forage should not be used for the production of grains or forage; and (4) suspected areas should be carefully studied as promptly as possible to determine definitely whether or not they produce toxic grain and forage.

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